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ORIGINAL ARTICLES.

EXSECTION OF THE SO-CALLED TARSAL CARTILAGE IN CASES OF CHRONIC TRACHOMA.*

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Illustrated with 8 Cuts.

SOME five years ago† I reported the results of the so-called Heisrath's operation in fourteen cases of chronic trachoma. As my subsequent experience has been equally and uniformly satisfactory, I am encouraged to bring the subject once more to the notice of the profession through this Association, particularly as I do not believe it has received the attention it deserves.

Let me repeat that this procedure is not, in my opinion, indicated in any of the recent or acute forms of trachoma nor would I advise it in any case where there is a reasonable prospect of early cure—not temporary relief merely—from any other form of treatment. When other remedies have failed and there is nothing before the patient but months or years of suffering—nothing but the "ups and downs" that characterize a deepseated trachoma, with its visual dangers and bodily discomforts from pannus, corneal ulcer, trichiasis and entropion, not to mention long continued and serious interruption of work—in many of such cases tarsal excision is

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†Removal of the Tarsus and Retrotarsal folds in certain cases of chronic Trachoma. *Annals of Ophthalmology*, 1898. p. 372.

certainly indicated. On the other hand, in the most advanced stage of the disease, in those cicatricial forms that have gone on to shrinking of the sac and in which there are probably few or no active trachomatous nodules in the tarsal cartilage, I do not think the operation is justifiable. Nor should it be resorted to if it is possible, as Gifford points out that it sometimes is, to remove, one by one, the discrete and scattered trachoma nodules from the tarsus itself.

Let me further declare myself in this important particular by more positive statements. Removal of the tarsus, in part or as a whole, is indicated in those long standing cases of trachoma, not amenable to other forms of treatment, in which the lids show trachomatous infiltration, with granula tion deposits in the connective tissue of the retrotarsal folds, whether the cornea be affected or not. If to these conditions be added thickening and enlargement of the tarsus itself, the operation is even more urgently indicated. Also, when there is evident disease of the folds, without apparent thickening of the cartilage, but the cornea is implicated, the operation should be done. A very important class of cases, from an operative standpoint, is that where with atrophy or cure of previously existing granulations in the tarsal folds there remain deepseated foci in the tarsus. In this troublesome and inveterate form of trachoma, whether the cornea has escaped or not, removal of the tarsus will give gratifying results.

The palpebral conjunctiva is rarely the only site of granular deposits in long standing cases of the disease. It is quite exceptional that the tarsus and submucous connective tissue escape. I believe that I am correct, therefore, in asserting that the simple method of excising the retrotarsal folds (long ago advocated by Richet and Galezowski) does not meet the requirements in such cases. The proposition is practically to remove the neoplasms that, in the later stages of this infection, are responsible for the destructive lesions of the disease. Why, then, should we remove a portion of these semi-malignant tumors and allow the others to remain? As long as there is reasonable ground for assuming that the activity of the trachoma colonies is confined to the conjunctiva and submucosa such procedures as *grattage*, the use of forceps, cauterization, excision of portions of diseased membrane, etc., are

of course, proper, and the method that I am about to describe is not intended for their relief.

Although it is desirable that the eye should be as quiet as possible before operation, I have not hesitated to excise the tarsus either in the presence of corneal ulcer, increasing pannus or during an acute exacerbation of the chronic disease. I have been satisfied with the results in these instances but have been careful to remove the stitches at as early a date as possible and to keep up constant disinfection of the eye while they are *in situ*.

A better understanding of the effects of the operation is gained by a reference to the muscular supply of the lids. According to Thomas Dwight and others the superior rectus,

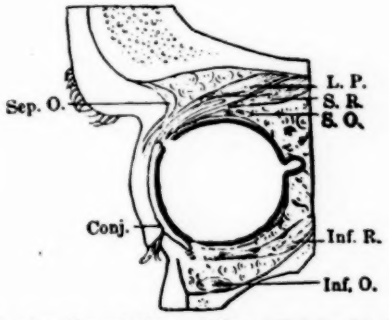

FIG. 1—(Dwight). Showing the relations of the orbital faschia to the tendons of the levator palpebræ, the superior rectus and the tarsus.

besides its insertion into the globe sends fibres not only to the top of the fold of conjunctiva, which is thus pulled up and back in harmony with the upward excursion of the eye but also to the top of the tarsus. Moreover "the levator broadens out into an expansion stretching across the whole orbit from one bony wall to the other, which, by its outer portion separates the greater lachrymal gland from the accessory portion below it. This expansion splits into two layers. The greater portion, consisting of involuntary muscular fibres, (Mueller's muscle), is inserted into the upper portion of the tarsus, while certain anterior fibres pass into or through the fibres of the orbicilaris to the skin of the lid. Their function is to draw the skin to the fold above the tarsus when the lids are opened. The expansion of the levator passing to the tarsus consists largely of

unstriped muscular fibres mingled with elastic tissue. This is connected with other involuntary fibres arranged transversely, the whole constituting what is known as "Mueller's muscle."* A somewhat similar arrangement exists in the lower lid, the tendinous expansion of the inferior rectus dividing into three layers, one of which is attached to the tarsus and taking the place of the levator of the upper lid in drawing the lid down in downward rotations of the globe and in opening the eye.

All the German authorities consider cocain a sufficient anesthetic for the operation. Kuhnt instills a 4 to 10 per cent. solution as a preliminary and then makes two or three subconjunctival injections of a 6 to 10 per cent. solution. I

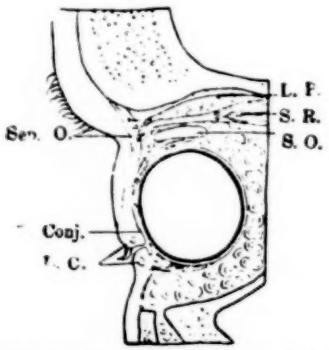

FIG. 2—From Dwight, showing the relations of the orbital fascia to the upper lid.

have not found this very satisfactory with my patients and I now invariably insist upon a general anesthetic. Not only is the operation a painful one (particularly when more than one lid is involved) but its success largely depends upon precision in placing the sutures and in other details difficult to carry out if the patient be restless or nervous.

The diagrams, from Kuhnt's work, illustrate the various steps of the operation, which I shall now proceed to describe.

When the operation is done, as it usually is, on the upper lid, the latter is everted so that the convex border of the tarsus is thoroughly exposed. This is now firmly grasped by two strong, toothed forceps at the junction of the middle

*Norris and Oliver. System of Diseases of the Eye. 1. 91. 92.

with the outer and inner thirds of the tarsal margin, and drawn firmly upward by the assistant standing at the patient's head. The junction of the palpebral and ocular conjunctivæ

EXCISION OF THE TARSUS.

FIG. 3—First act. Eversion of the upper lid with forceps (Kuhnt).

is now fully exposed and may be readily examined. Following as nearly as possible the margin of the diseased area, an incision is made from the outer to the inner canthus through

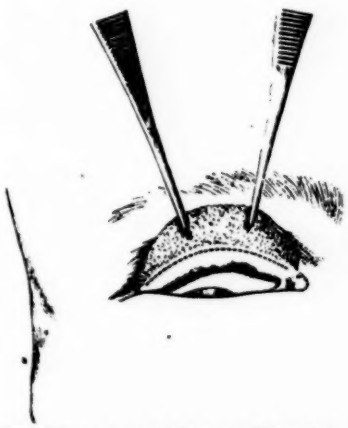

FIG. 4—Second act. Complete eversion of the upper lid and exposure of the retrotarsal folds. First incision along the dotted lines (Kuhnt).

the conjunctiva only. Unless, in consequence of previous mechanical treatment, the conjunctiva is bound down to the underlying tissues, the wound will gape and the fibres of Mueller's muscle may be recognized. Three stitches should

now be passed through the bulbar margin of the incision, care being taken to include only the conjunctiva and a few fibres of the submucosa. If more than a mm. in width of conjunctiva is included in the sutures, small symblepharon folds may form opposite each stitch, and if too deeply inserted there will be a noticable dragging on the lid edges, as occurred in one of my own early attempts.

A word as to the sutures. My assistant, Dr. Frank Brawley, has prepared for me a modification of the black silk (preferably No. 2 black braided) which Worth advises in his advancement operations. I have used them for the past year with great satisfaction in all operations that involve the con-

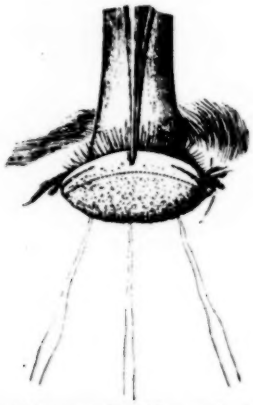

FIG. 5—Third act. Second incision near the lid margin, after placing of the sutures in the upper border of the first wound (Kuhnt).

junctiva and I warmly advocate their employment in the procedure about to be described. The silk is first wound upon ordinary glass microscopic slides (for convenience of handling) and sterilized by boiling 30 minutes. It is then dehydrated by immersion in absolute alcohol for 10 minutes and the drying process assisted by holding the slides a few feet above a Bunsen burner flame for a few additional minutes. The slides of silk are then dropped into a jar of paraffin containing 25 per cent. of vaseline, where they remain until used. Each time they are used the jar containing the silk is resterilized by heating, an end of suture is drawn out of the jar and the excess of wax is "stripped" off the required suture lengths by drawing it through sterile gauze held between

the thumb and finger. The threads are now somewhat stiff yet flexible, are easily threaded, never "kink" and slip through the tissues with the minimum amount of friction and traumatism and do not readily tear out of the tissues in which they are placed. Moreover, knots made in these threads are much less likely to irritate and abrade the cornea or bulbar conjunctiva. Once introduced through the lower wound margin they should be allowed to hang down over the globe (see the diagram) and to rest on a sterilized towel placed on the cheek. After the sutures have been thus placed the bulbar conjunctiva should be separated from the globe a distance

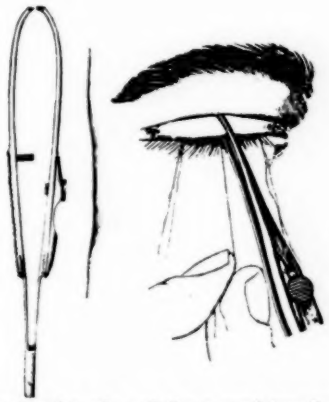

FIG. 6—Fourth act. Bringing the edge of the wound together after tarsal excision. Estimating the proper place to enter the needles below. Forceps used in operation (Kuhnt).

of 3 to 5 mm. from the edge of the wound. The forceps may now be removed from the convex border of the tarsus and the lid margin be grasped at its middle point, a horn spatula being passed behind the everted lid, as shown in the diagram. A second incision, running the whole length of and parallel to the lid edge, is now made as nearly as possible in the healthy conjunctiva. Sometimes this will be three, sometimes even five mm. from the palpebral border, the intention being to remove as little of the unaffected mucuos membrane as possible and so to leave as large a portion of the central conjunctiva area as is consistent with the needs of the case. The spatula may now be removed, the assistant drawing the lid upward and backward with one or two fixation forceps. The operator then seizes the tissues at the nasal junction of

the two incisions and with scalpel and scissors slowly excises conjunctiva and tarsus, carefully avoiding the orbicularis and Mueller's muscle. At this point the anesthetic may be removed, and time allowed for the bleeding to cease. I have

FIG. 7—First act in the operation for removal of the tarsus without sacrifice of a fairly healthy conjunctiva (Kuhnt).

not been much troubled with hemorrhage, although some small branches of the arterial supply may have to be twisted.

The conjunctival sac should be thoroughly irrigated and the lips of the wound brought together. To secure asatisfactory result one must be particular to place each suture in

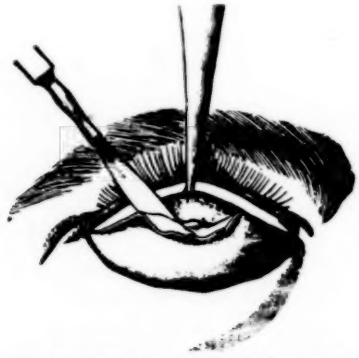

FIG. 8—Second act in the subconjunctival excision of the tarsus (Kuhnt).

both wound margins so that it will be exactly oppose its fellow when the eye is closed. It is also requisite that the bulbar conjunctiva should not be too much put upon the stretch. The middle suture should first of all be tied with a single knot and it is wise to make certain, by closing the lid, to ascertain whether the precaution just mentioned has been taken

before the final knot is tied. I would advise the operator to allow the patient to recover from the anesthetic sufficiently to enable him to determine accurately whether the palpebral movements are sufficient and to be sure that there is no irregularity visible at the lid margins. If the interpalpebral space is the same, both with the eye open and shut as it is on the opposite side, and if the lid margins have a regular outline, all is well and the threads may be cut off close to the knots.

As a rule there is little subsequent pain, and very little reaction. The *after treatment* is simple and need not interfere with the attention properly demanded by the presence of corneal ulcer or other complications. On the whole I find gentle irrigation of the sac four or five times daily with warm boric acid solution, followed by the instillation of warmed and steril vaseline, is grateful to the patient and acts very nicely. The vaseline keeps the sutures soft and serves to protect the cornea. I apply a light bandage over both eyes and order the patient to keep quiet, but do not insist upon his remaining in bed. The sutures are removed on the fourth or fifth day. In a week or ten days the wound is usually quite healed but the sac should be subsequently examined for the presence of granulation tissue or irregular wound margins. These are best clipped off or trimmed with scissors.

It is advised by some operators that no stitches be used, owing to the possibility of corneal lesions. For my own part I have not witnessed these untoward results when the precautions I have mentioned have been taken, and I am sure that healing occurs sooner and the cosmetic effect is better when sutures are employed.

I do not see a great many cases of chronic trachoma, either in my private practice or in my dispensary work, and I have operated on only eight additional cases since my last report, but these have been so satisfactory to me that I take the liberty of reporting several of them here, choosing those instances that seem to me to illustrate the majority of the conditions in which, in my opinion, the operation should be done.

The most important result of this operation is the relief given to the irritative symptoms of the disease. Very shortly after the removal of the stitches we notice the subsidence of

the photophobia, the lachrymation, the foreign body sensations and the local discomfort that accompany chronic granular lids, even when there is no ulceration of the cornea or no acute conjunctivitis present. Pannus is always lessened and may even disappear, and as a direct consequence of this the sight is much improved. In one very severe case of corneal infiltration, referred to in my previous communication, where the visual acuity had fallen to $^1/_{10}$ it rose to $^2/_7$ within three months after the tarsal excision. The asthenopia generally exhibited in the better eye of a case of chronic unlateral trachoma is wonderfully improved, and, strange to say, in not a few cases those common sequels of chronic trachoma, entropion and trichiasis, are decidedly less marked than before the operation. Finally, when we have to deal with recurrent ulcer of the cornea, a cure of the abnormal conditions behind it generally prevents a return of the disease.

The objections that have so far been urged to the removal of the tarsus are:

(1) That *ptosis* is likely to follow the operation owing to section or exsection of the levator palpebral superioris. I have never seen a single instance of this sequel in the twenty-two cases in which I have myself operated on nor in patients under the care of others. Owing to the relief from spasm of the orbicularis palpebrarum (always more or less present in all forms of chronic trachoma) the patient invariably opens his eyes to a greater extent than before, and I have not witnessed and do not expect, as the result of the removal of the tarsus, any diminution in the size of the interpalpebral fissure or in the power of the levator muscle. I believe the reason is that the attachment of the muscle is not to the tarsus alone.

(2) The same denial I would like to enter as to the probability of *entropion*. The fact that a shrinking and diseased tarsal cartilage is one factor in the production of lid edge incurvation accounts for the fact, sufficiently noticed by Kuhnt and seen in several of my own cases, that after the operation the previously incurving cilia gave no further trouble. In one instance, referred to in my first paper on the subject, and in another more recent one where epilation had been practiced for long periods, the eyelashes no longer required to be removed.

(3) *Ulcer of the cornea* has been noted after this operation, and it seems reasonable that the rubbing of the stitches and knots over the eyeball might produce an abrasion followed by infection. I have never had such an experience and do not think that if the directions for the operation, especially with the use of specially prepared sutures are followed, it is not likely to happen. Moreover, when one remembers that serious corneal ulcer may occur at any moment in these very cases, with or without treatment, it seems hardly proper to lay this accident at the door of the operation.

(4) The one complication to be avoided in Heisrath's procedure is the production of irregular, symblepharon-like folds in the region of the sulci. Unlike the alleged dangers just discussed, which are more imaginary than real, this is an accident which *may* happen to the careless operator. When it does occur it is very annoying to the patient, inasmuch as the ocular excursions are attended by dragging, drawing sensations, and in nervous subjects are exceedingly irritating. For relief of them it is best to dissect back the symblepharal attachments and implant mucous membrane or skin grafts to cover the denuded surface.

The following cases may serve to further illustrate the contentions of this paper:

Case 1.—Sister V.'s eyes were infected from a patient in a St. Louis hospital in 1893. She was treated during the following year by Dr. Alt and when I saw her, two years after, the disease had assumed the usual chronic form of the follicular type; the granulations were not exuberant, but corneal complications were frequent. The patient was unable to use her eyes and suffered much from foreign body sensations, headache and photophobia. She had numerous small nebulæ corneæ and a superior pannus on both sides. She came to me chiefly on account of pin-point ulcerations on the left cornea. I treated her with varying success by all sorts of methods and remedies for nearly a year. She had many relapses in the meantime and her visual power slowly declined. Finally, in December, 1897, I persuaded her to have both tarsi removed from the upper lid. This was done and the patient slowly recovered. To-day she has, with correction of her myopic astigmatism, $^2/_7$ vision in the left eye and a little better in the

right eye. She reads words of Jaeger 1 with the right eye and of Jaeger 4 in the left. More important, her eyes are quite comfortable since the spring of 1898, when she had one corneal ulcer due, probably, to a number of infected follicles, which were not removed at the time of the operation and which I treated subsequently with lapis divinus.

Case 2.—Mr. A. N. was attacked by inflammation of the lids in 1882 and until comparatively recently has been under treatment in France and in this country. I saw him in May, 1897, and found him suffering with well marked chronic trachoma in both lids, pannus in both eyes, a beginning staphyloma left (for which Galezowski had done an iridectomy), there were granulation masses in the left upper and lower folds, upon which I used the forceps. Then began a long, weary attempt to get rid of the disease. The patient was treated all summer. When I saw him in September, 1897, the secretion and granules were less, but he had had several pin-point ulcers in both eyes and was much discouraged. I did a Heisrath's operation on the left or better eye ($V=^{7}/_{200}$) and later on the R. The right lens becoming cataractous and the corneal staphyloma increasing, I removed the lens, with improvement. He began to get better and to-day is practically cured of his trachoma, but bears the scars of the contest. In left eye V with correction of $A=^{20}/_{70}$—and words of Jaeger 4 at 10 cm.

Case 3.—Oct. 21. Celia H., school teacher, had to relinquish her occupation on account of a chronic eye inflammation which affected both eyes in 1897. In September, 1900, I saw her, when she had a well developed follicular trachoma, affecting both lids of both eyes. V. R.=fingers at 1 meter: V.L.$=^{20}/_{30}$—. There was total pannus R., by far the worse eye. Treatment was followed by improvement left but very little R. Heisrath's operation done R: many granulations found in the wound, but all lid movements good. Treatment with lapis after use of forceps in the left affected a cure. Patient returned a few weeks ago to say she had resumed school six months after the operation perfectly well. With correction V.L.$=^{20}/_{20}$. V.R.$=^{20}/_{70}$. There are nebulae plainly to be seen on R. cornea but the operation scar is insignificant and there are no symblepharal folds.

Case 4.—Maggie Driver, age 27, Versailles, Mo. Has had trachoma in both eyes for 12 years, with repeated exacerbations, corneal ulcers, pannus, etc. Father, mother and 11 brothers and sisters had disease. First saw her Nov. 7, 1902, when she had an acute attack of trachoma beginning four weeks previous. A well marked pannus present with some staining of left cornea with fluorescein. Right eye not so bad. Trichiasis both eyes. Right lower lid has been operated upon for entropion. Marked improvement under 20 per cent. argyrol used three times a week in office and a 5 per cent. sol. three times a day at home. Cold applications every 2 hours followed by boric acid lotion.

Jan. 7-8, 1903. Tarsus of R. eye removed.

Jan. 7-15. Tarsus L. eye removed.

Made an uneventful recovery.

LV. $^{5}/_{200}$. RV. $^{11}/_{200}$.

The excision of a part or the whole of the so-called tarsal cartilage, without sacrificing the overlying conjunctiva, is an operation suggested by Kuhnt in those cases where the disease has practically died out in the mucuos membrane but is active in the submucuosa and tarsus. I have had no experience of the method, although the *technique* presents no difficulties and the operation seems rational.

DISCUSSION.

Dr. Oscar Dodd, Chicago.—What effect has this on the cul-de-sac? Perhaps I did not hear the paper as distinctly as I should, but I did not get the exact explanation as to that part.

Dr. Albert E. Bulson, Jr., Fort Wayne, Indiana.—I have been much pleased with this paper for the reason that it advocates an operation which will certainly give relief to a large number of cases suffering from the ill-effects of a thickened and inverted eyelid following trachomatous inflammation.

I wish to digress a little by reporting a case operated in a similar way to that described by the essayist. After repeated failures to secure satisfactory results in an aggravated case of entropion (the Hotz operation and several other measures being resorted to), I conceived the idea of removing a portion of the thickened cartilage which I thought in a very large measure responsible for the continued irritation of the eyeballs and prac-

tical loss of vision in both eyes from pannus. The operation was decided upon as being a measure that would produce no harm if it did not produce relief, for there was everything to gain and but little to lose in the treatment of the case. Under a general anaesthetic the conjunctiva was dissected up, and practically the entire tarsal cartilage removed, only a small part of the upper portion being allowed to remain on the theory that it might act somewhat as a support for the remaining tissues. The case went on to recovery, and I had the satisfaction of seeing the pannus clear up to a certain extent and vision improved so that the patient could see to get about without assistance, whereas before, his vision was so poor that but little more than shadows were seen. Some ptosis remained, but this was not sufficient to prevent vision, and the patient was so thoroughly satisfied with the results that he made no complaint regarding the slight drooping of the lid.

I am convinced that the operation is beneficial in a certain class of cases, but think it should be reserved for those obstinate conditions in which inversion of the lids continues in spite of all other treatment usually employed.

Dr. Adolf Alt, St. Louis, Mo.—I would like to ask what effect the removal of the tarsus has on the remaining granulating tissue. This operation leaves a strip of conjunctiva along the upper edge of the tarsus and there is undoubtedly some trachoma in the tarsal fold, although there was not in the pictures. Is there no further growth of the trachoma? I have never seen a case yet in which I felt obliged to resort to this operation. It seems to me Dr. Prince read a paper on the same subject at our meeting some five or six years ago in Chicago. If it is of so much benefit we will have to adopt it as a legitimate measure.

J. B. Worrell, M. D., Terre Haute.—I never had occasion to make the operation. I remember some years ago having seen a case in which the tarsus had been excised but do not know the technique in the case. Last year I saw a very unfortunate case in which the operation had been done and the patient had no lid left. Whether it was for the removal of trachomatous tissue or not, I cannot say. The problem was to restore the lid, and to a certain extent it was accomplished. I rise to ask two or three questions; in the operation, do you remove all the tissue between

the two incisions, afterwards stitching the two edges together? (Dr. Wood.—Yes, where possible.) What is the effect of this upon the cul-de-sac? Would it reduce that to such an extent that it would limit the movements of the eyeball? Do you remove the tarsus entirely, leaving only the muscular tissue of the lid? (Dr. Wood.—Every bit of it.)

Dr. Casey Wood (closing discussion).—The main question in cases of inveterate trachoma is, shall we go on treating them by the usual ineffective method, or try to eradicate the foci of the disease itself? So far as the formation of tears is concerned, the removal of the tarsus has not made any difference at all. In a few cases there was a complaint of "dry eye;" and one may easily believe that in removing a large amount of conjunctiva a considerable secreting area disappears; but it must be remembered that xerosis is frequently present in old cicatricial trachoma so that we may usually refer these dry sensations to the ravages of the disease. Except in one case, the author has never noticed any limitation of the upward or downward excursions of the eyeball. In simple excision of the folds of transmission for this disease, an operation often done and described by French authors and those of the German school, no mention whatever is made of subsequent limitation of the globular movements. Would it be an insuperable objection to the operation, even if it did limit these rotations? External examination rarely shows that an operation has been done. In the majority of instances it is only when one attempts to evert the lids or to examine the condition of the folds that the absence of the tarsus can be noted at all.

The effect of tarsal excision on the remaining conjunctival tissue is very slight. However, the trachomatous tissue that remains can always be reached by remedies and I have not found any difficulty in so treating it, because it is superficial and can be reached by ordinary methods. But the nodules that are imbedded in or attached to the tarsus cannot be reached by mild means, and that is the reason for the operation. Our interest is to get rid of these active foci of the disease, which lie deep in the tarsus itself.

PARALYSIS AND PARESIS OF THE MUSCLE OF ACCOMMODATION.*

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IT is the endeavor in this paper to consider the paresis and paralysis of the muscle of accommodation as distinct and separate pathologic entity; and, to lay before the members sufficient valuable associated data so that our attention might more often be attracted to this particular lesion than has hitherto been our custom. Furthermore, it is intended to confine the consideration mainly to the so-called peripheral type. Topographical anatomy and physiological experiments warrant us to assume that the function of accommodation is an independent one with reference to the other functionings of the eye as such (Hess, 1902; Mauthner, 1899; Hensen and Voelckers, 1878). This does not make it necessary to exclude the so-called con-commitant associated acts of the iris and extraocular muscles.

The first correct conception or description of either paralysis or paresis, was given by Wells[1] (1811). He also knew full well the action of mydriatics on the power of accommodation. He did not, however, know of the various nerve centres concerned in the function of accommodation or pupillary reactions. This remained to be deciphered by Helmholtz[3] (1853), Bruecke[2] (1846), Huebner (1872), Mannhardt[4] (1858), Hensen[5] and Voelckers (1868).

Prior to these days, paresis and paralysis of accommodation were often confounded with rapidly oncoming presby-

*Read before The Academy of Ophthalmology and Oto-Laryngology at Indianapolis, Ind., April 9, 1903.

[1]Wells, P. Philos. Trans., cl., p. 378, London, 1811. (See Donders Accom. and Refraction, foot note p. 597).

[2]Bruecke, E. Ueber den Musculus Cramptonianus u. d. Spannmuskel der Chorioidea; Mueller's Archiv., 1846.

[3]Helmholtz, H. Ueber d. Accommodation d. Auges; Archiv. f. Ophthal. (Graefe), 1855, 1. 2.

[4]Mannhardt. Bemerkungen ueber d. Accommodationsmuskel u. ueber die Accommodation; Arch. f. Ophthal., iv, 1858.

[5]Hensen and Voelckers. Experimentaluntersuchung u. d. Mechanismus der Accommodation; Kiel, 1868.

opia (Himly[6]). Before the days of cerebral localizations and the physiology of presbyopia, it was not at all surprising to make this error, as in both conditions the near point recedes, and the far point remains about the same as before. Not only was it confounded with presbyopia but with many of the present day amblyopias and amauroses (Nagel, 1866 and Donders, 1866, mention this fact in a foot note). A dilated pupil is frequently a pertinent objective symptom in paresis and paralysis and at the same time accompanies many of the amauroses. It was, therefore, not at all strange for the older writers to often confound the two conditions. Especially as we now know paresis and paralysis of the muscle of accommodation is not such a clinical rarity.

From Himly's time (1811) down to Donders (1864) very little was said about these conditions of the ciliary muscle. In 1860, Donders[7] gave the first accurate description of this ciliary affection as a sequel of diphtheria faucium (from an epidemic then in Paris). Since then, it has received more or less attention, but it was always associated with diphtheria and what is now called ophthalmopegia interna.

Not knowing that the centre of accommodation is one independent of the other eye centres, they never looked for paralysis or paresis as occurring unassociated with other intra- or extra-ocular paralysis. That they did not recognize this condition of the accommodation in ophthalmoplegia totalis, is evident from the writings of a certain Brunner[8].

Though the muscular mechanism of accommodation had long been known (Brücke, 1846; Helmholtz, 1855), and some of its errors recognized, yet they failed to locate the lesions and centres. Neither did they interpret the occurrence of the lesions correctly. The condition was not even

[6]Paresis and par. of accom. at this period was called "ciliary amaurosis" (Walther, Jour. von Graefe and Walther, Bd. 111, 22; "amblyopie presbytique" (Sichel Annal. d'Oculist. 1853).

[7]Donders. Paralytische Sympt. nach Diphtheria faucium; Archiv. f. d. holland. Beitrage z. Nat. u. Heilk., 1860, Bd. 11, 453. (note) Faure in the L'Union Med. 1857, mentions diph. par. in six cases and says that Trousseau, Bretonneau and Blache also observed it. Prior to this time, it was called "amblyopia" (Guimer, 1838; Ozanam, 1835).

[8]Brunner, C. De paralysi musculorum oculi nonnulla (Berolini, 1850, p. 10. He first used the term Ophthalmoplegia Totalis, but he does not speak of any condition wherein accommodation alone was affected and the other muscles intact.

fully appreciated as late as 1878, as is evidenced by Hutchinson[9]. In an article of Ophthalmoplegia Interna, he prematurely places the lesion in the ciliary ganglion. To-day, we know that this ganglion has very little to do with accommodation whatsoever as a separate centre. The researches of Hensen and Voelckers have shown beyond a cavil that Hutchinson's assumptions were erroneous. Hutchinson evidently was not aware of the experiments of Hensen and Voelckers[10] (1878), Hulke and Schweigger[11] (1858).

At the present time, we accept the facts regarding the centers of the accommodation and pupillary reaction as laid down by Huebner, 1872; Voelckers and Hensen,[12] 1878; Kahler and Pick[13], 1881. These have demonstrated a series of centers in the nucleus of the third nerve, which are associated with different functions. These centers lie partly in the posterior part of the third ventricle and partly beneath the corpora quadrigemmina, in the floor of the aqueduct of Sylvius. They lie directly behind one another. That portion of the nucleus of the third, in the posterior extremity of the floor of the third ventricle just in front of the opening of the aqueduct, causes ciliary muscle contraction; a little farther back in the aqueduct is the iris contraction centre; the centre for the external ocular muscles is the next. From this arrangement, it can be said that in the loss of their functioning, we have:

(a). Loss of the power of accommodation.

[9]Hutchinson. "Ophthalmoplegia Interna;" read before the Roy. Med. and Chir. Soc., 1878. H. was the first to clinically speak of the terms "ophth. interna and externa." Graefe in 1868 uses the same term in speaking of analogous conditions. Eulenburg, 1871, speaking of similar affections of the third nerve appropriately applies the term "progressive" (very fitting for nuclear paralyses).

[10]Hensen and Voelckers. Vide general references, No. 2.

[11]Schweigger and Hulke in 1858 demonstrated ganglion cells in the front of the choroid and ciliary region and associated them with the action of the iris. Graefe-Saemisch Handb. d. gesammt. Augenheilk., vol. 1. 1858.

[12]Hensen and Voelckers' (v. gen. ref.) arrangement: 1, Accommodation. 2, Sphincter iridis. 3, Rectus internus. 4, Rectus superior. 5, Levator pal. 6, Rectus inf. 7, Oblique inf. 8, Trochlearis.

[13]Kahler and Pick's arrangement (Prager. Ztschr. für Heilk. Bd. 11, 4, pp 301): 1, Accommodation. 2, Iris. Medial: 3, Rect. sup.; 4, Rect. ext. Lateral: 5, Levator pal.; 6, Rect. sup.; 7, Oblique inf. 8, Trochlearis.

Experiments on cats, dogs and monkeys have verified this classification.

(b). Loss of the pupil contractions.

(c). Paralysis of the muscles supplied by the third nerve. Often the fourth and sixth are involved because their centres are contiguous to the third. However, an association of "a" and "b" constitutes an ophthalmoplegia interna. This ophthalmoplegia can be complete or incomplete, especially if we incline to Mauthner's view of the nuclei.[14]

Furthermore, the blood supply (Huebner[15], 1872; d'Astros and Alezias[16], 1892) to these intrinsic centres, as laid down by these experimenters, will add material weight that one of these centres for the various functions of the third nerve can be affected without implicating the other two, in any shape, form, or manner. You can have a paralysis or a paresis of the extra-ocular or intra-ocular muscles, separately or combined; or, only one muscle in either group. But, it may be asked why it is that the iris and ciliary centres are more often involved than the others. No satisfactory reason can be assigned for this.

According to the foregoing, Hutchinson's theory that the ciliary ganglion is the nucleus for ophthalmoplegia, is evidently untenable. The ganglion would have to be divided in a manner like the third nucleus and this we know is not so.

[14]Mauthner. Ich bin auf Grund d. klin. Beobachtungen d. Ansicht dass d. Nerven f. d. Augenmuskulatur jedes Auges saemmtlich ihre Kerne auf d. gleichnamigen Seite haben u. d. daher d. einseitige totale Ophthalmoplegie einfach durch d. reihenweise Erkrankung d. gleichseitigen Nervenkerne bedingt wird. Die Lehre von den Augenmuskellæhmungen, 1889, S. 368.

[15]Huebner. Zur Topographie der Ernaehrungsgebiete der einzelnen Hirnarterien; Centrbl. f. d. med. Wissensch. 52, S. 818, 1872. H. clearly shows that the circulation of the brain cortex and the base are entirely different. In the former innumerable anastomoses occur with an extensive ramification; in the latter, the vessels after a short course dip into the substance of the brain and supply the immediate surrounding areas without anastomosing. In other words, we have typical end arteries in the basal circulation and not in the cortical. They are end arteries according to Cohnheim. Each supplies a distinct, though small brain area; and, the supply of the third nucleus is no exception. The vessel supplying the nuclei of the iris and the accommodation, is the ramus communicans posterior and does not supply any other part of the third nucleus.

[16]d'Astros and Alezias dispute Huebner and say the blood supply comes from the posterior cerebral of the basilar and not directly from the basilar. It is called the arteriæ nucleoli oculomotorii. (A Les artéres nourricières des noyaux du motaire oculaire commun et du pathétique; Soc. de Biol., Juni 1892).

Experiments uphold Hensen and Voelckers but not Hutchinson. Again, it is very reasonable to suppose that a ganglion of the sympathetic group and not of the cerebrospinal, in addition to being removed from the nerve centres, has more to do with "intimate association" of closely allied automatic functions than with their differentiation from one another (W. A. Sturge, V. vf). Spec. ref. No. 8.

It may be stated as a fact (experiments in the past and the present bear out the assertion) that upon the removal of the ciliary ganglion[17] and the stimulation of the cervical sympathetic[18], the pupil still dilates to a considerable extent. The reverse holds true also. Furthermore, the pupil dilates when both mentioned ganglia are excised, and atropine is instilled or a peripheral irritation made. When eserine is instilled under these conditions, the catoptric images change. These points show that the iris must be supplied with dilating fibres derived from a different source than from the ones already noted. These accessory fibres are furnished by the ramus ophthalmicus of the fifth nerve. They enter the eye along with the optic nerve, upon the superior portion and external to the optic sheath. Division of the ramus causes greater pupillary reaction, i. e., contraction, than upon sectioning the ciliary or cervical ganglia. Hence, the iris must receive dilating fibres independent of the ciliary or cervical ganglia.

As the iris under these various conditions still responds to eserine and atropine, and as these latter only affect nerve terminal centres, we are compelled to at least think of the ganglionic centres in the chorioid, iris, and ciliary body as announced long ago by Hulke[19], and Schweigger[20] (1858), and Meyer[21] (1893). These plexuses possess the autonomy of nerve centres and hence will assist us in explaining many pupillary phenomena otherwise inexplicable.

Accepting these facts regarding the peripheral and central nerve centres for the accommodation and iris reaction, it re-

[17]Adamück. Centralbl. f. d. med. Wissensch., No. 28, 1876. Hensen and Voelckers, Loco citato.

[18]Hulke. See gen. ref.

[19]Schweigger. Loco citato.

[20]The writer has many times noticed this condition while experimenting with these ganglia in reference to glaucoma.

[21]Meyer. Zur Kenntniss zum Bau der Iris; Biolog. Untersuch. Neue Folge. V., 1893.

mains to be demonstrated whether the sympathetic nerve has any connection with the accommodation. It is an axiomatic fact that the motor oculi is the nerve of active accommodation and ciliary muscle contraction. Also an axiomatic fact, that a paralysis of a part of the third nerve causes a paresis or paralysis of the act of accommodation. In vain have experimenters tried to prove the sympathetic nerve supply for it. The sympathetic fibres were supposed to be inhibitory accommodation fibres[22]. It would be truly convenient to have this inhibitory supply in explaining many apparently unconnected symptoms regarding lesions in these various centres.

Morat and Doyon, upon cutting the sympatheticus and stimulating the cut end, noticed an enlargement of the catoptric images and concluded therefrom that the nerve acted as an inhibitor and assisted in the accommodation for distance. Upon repetition of this experment by others[23], it was found not to prove true. Langley and Anderson emphatically deny Morat and Doyon, and are upheld by Hess[24], Rœmer and Dufour[25].

These fibres were supposed to call forth a decided flattening of the lens through their action on the inner layer of the ciliary muscle. Though there are two distinct muscle layers, an outer and an inner, they act as a single muscle[26] (Bruecke in Mueller's Archiv., 1846, p. 370). As far as we know their contraction causes a drawing forward of the ciliary body and processes, a relaxation of the zonnula of Zinn, a swelling

[22]Morat and Doyon. Le grand sympathétique nerf de l'accommodation pour la vision des objets éloignés; Compt. Rend. de l'Acad. des Sc. and Archiv. de Physiol., III, 507, 1891.

[23]Langley and Anderson. On the mechanism and movement of the iris; Jour. of Physiology, XIII, 6, 1892.

[24]Hess. Arbeiten aus d. Gebiete d. Accommodationslehre; Graefe's Archiv. f. Ophth. XLIX, 2, 1899. Graefe's Archiv. f. Ophth. XLII, 1896, and, XLIII, 1897.

Hess. Bemerkungen zur Accommodationslehre; Centbl. f. prak. Augenheilk., July, 1899.

[25](Dufour and Rœmer assisted him (Hess) in this work).

[26]That the ciliary body was muscular, was a well known fact to Kepler in 1611; Eustachius in 1722, even had a diagram thereof. Porterfield (1759), Morgagni, Briggs and others knew of the action of the ciliary muscle. Zinn, however, denied that they ever knew the existence of the muscle. In 1846, Bruecke described the "tensor choroideae;" about this time it was also described by Bowman. In 1856, H. Mueller described the inner muscular layer of the ciliary body.

of the anterior surface of the lens and a sinking of the same. Hensen and Voelckers needle experiment[27] absolutely proves that there is no muscle in the eye purposely to assist in focusing it for the distance. The catroptric images also seem to disprove this assumption.

Having determined that the ciliary muscle, the third nerve, and a separate central nucleus controls the accommodation, we may ask what relation the action of the iris sustains to this function. The contraction of the pupil during accommodation is the oldest known symptom in connection with it (note).

It was long thought that accommodation depended on the contraction of the iris. However, Koster[28], and before him Weber and Hess[29] had determined the pupil contraction to be only associated with convergence and not accommodation. Tscherning[30] regards the accommodation pupil contraction as a mechanical manifestation. At one time it was thought that the contraction of the iris was due to the amount of blood in it during the act. This idea is untenable, as it has never been demonstrated. The blood pressure in the iris and the ciliary body depend upon their inherent contraction activity as is seen in any other muscle. The contraction of the iris is not an essential factor in accommodation, but only an associated physical phenomenon.

[27]H. and V's., experiment. They inserted needles into the equator of an enucleated eye (and earlier into the living animal) then electrically stimulated the ciliary body region and watched their motion as follows: One needle advanced forward, showing advancement of the chorioid; the one through the ciliary did not move; one very near the macula appeared to remain perfectly quiet (latest references to this experiment is made by Hess, in Graefe-Saemisch, 1902).

NOTE.—Scheiner (1619), Morton and Haller (1769), tried to explain all accurate accommodation by the action of the iris. It was Hering and Donders who demonstrated the synchronous action of pupillary and ciliary contraction during accommodation, but they did not say whether the pupillary contraction was a reflex act or not during accommodation, which it truly is.

[28]Koster. Bemerkungen zu den Versuchen von Hess ueber d. Accommodation Archiv. f. Ophthal. XLVII, 1., 1898.

Koster. Ibidem; Entoptische Beobachtungen; Archiv. f. Ophthal. XLVI, 1., 1898.

[29]Graefe-Saemisch Handb. d. gesammt. Augenheilk., Bd. VIII, Kap. XII (1902).

[30]He demonstrated a dilatation and a contraction of the pupil on the cadaver by inserting a hypodermic syringe and gently drawing the piston forward and backward.

Some facts have been set forth which warrant us to speak of paresis or paralysis of accommodation as a separate manifestation. From the foregoing we can look upon this affection of the accommodation at times as independent of internal or external ophthalmoplegia. It matters little whether you accept the Young[31]-Helmholtz or the Tscherning[32] theory of accommodation, the symptoms are the same.

With a paralysis or a paresis of accommodation, it is then not so strange to have the other so-called associated functions involved to a certain degree. The loss of the function of any one of these three materially influences the extent of action of the remaining two. This difference in the functioning of the other two may, in many instances, be only apparent. (It is not deemed necessary to enter upon the actual mechanism of accommodation, only a brief outline[33] is needed).

From a standpoint of practical symptomatology, there is very little difference between a paralysis and a paresis[34]. Yet, pathologically often a great difference is manifested. A paresis is frequently a partial functional loss or inability, with no demonstrable lesion, excepting those perhaps peculiar to exhaustion[35]. A paralysis on the other hand is very often the

[31]Young demonstrated accommodative changes to take place in the lens by experiments now renowned (On the Mechanism of the Eye, Phil. Trans. 1801).

[32]Vide gen. ref. No. 6.

NOTE.—Descartes, 1637, was the first to associate convexity of the lens with accommodation (Trans. de homine). Mauthner gives credit for this to Scheiner in 1619 vide M. Augenmuskellaehmungs Lehre, 1889.

[33]The act of accommodation ensues as follows: 1. Stimulous from central portion of third nerve nucleus. 2. Contraction of ciliary muscle. 3. Slight forward traction of chorioid and perhaps of retina. 4. Relaxation of the Zonula. 5. Increase in the ant. convexity of lens, with lessened vertical and increased antero-posterior axis. 6. Sinking of the lens. 7. Decrease in the depth of ant. chamber. 8. Trifle forward movement of iris. 9. Perhaps the vitreous also moves forward. The associated functions are: 1. Contraction of iris. 2. Convergence, with a trifle turning downward of the eye.

[34]An exhaustion of a muscle is often similar to a paresis. At times they are not to be differentiated.

[35]Paralysis. The suspension or abolition of functional power, especially in the nervous system, in which case there is a temporary or permanent loss of the power of motion or sensation or both, in the parts supplied by the affected nerve.

Paresis. An incomplete paralysis, especially when not associated with any demonstrable organic lesion—limited to motion and not to sensation (Foster).

result of active pathologic changes—peripheral, central, or both.

We must differentiate between an active and a passive variety of paresis or paralysis of the act of accommodation. That is to say, active when the ciliary muscle or nerve function is involved; passive when the lens or the zonula alone are implicated. In the active, we distinguish a myopathic and a neuropathic type. Again, the active type is either a peripheral, a central nucleus, or an orbital lesion. The paresis is more often a peripheral lesion (myopathic) rather than a nuclear or orbital; while the paralysis is apt to be a nuclear or a peripheral nerve lesion.

The changes in the passive portion yield the same symptoms as those in the active part. Therefore the two conditions are easily confounded[36]. Any change in the passive part, either in the lens or zonula, is demonstrable by the shrinking of the total range of accommodation. However, not every ciliary muscle paralysis is evidenced by a receding near point and a shortening of the accommodation range.

Either the active or the passive variety may appear as a separate manifestation, unassociated with any other symptom. But as a symptom itself, only the active variety can be associated with an ophthalmoplegia interna, externa, or totalis. In the former two it may be absent, but in the totalis it must be present.

To make the diagnosis of paresis or paralysis of accommodation, we have to take into consideration the actual range of accommodation and to determine the range of the accommodation which is characteristic of that age of the patient in whom the paresis occurs. This latter is dependent on the passive part of the accommodation. On account of the increased hardness in the lens with age, so in proportion does the contraction of the ciliary muscle become optically less evident. In old age the effect of this contraction is entirely latent. Hence it follows that any ciliary muscle contraction is more readily recognized in the decreased accommodation range in the younger individuals, as they have very

[36]Accommodation paresis is not the same as ciliary paresis. This is evident in presbyopia, which is an accommodation paresis but not a ciliary paresis. A normal eye can have the normal range of ciliary contraction, yet the altered lens will not respond.

little that is latent. In the aged,[37] say 70, the ciliary muscle can be absolutely paralyzed and yet no change in the range of accommodation or recession of the near point takes place.

The objective proof of a ciliary muscle paresis or paralysis is only demonstrable when it is greater than the range of latent accommodation. In other words, it must reach such a degree that by the utmost contraction of the ciliary muscle a complete relaxation of the zonula is not obtained. Again, any decrease in the manifest accommodation (peculiar to the age of the patient at the time) is evidence of the presence of a ciliary paresis or paralysis. But on the contrary, not every ciliary paresis or paralysis is evidenced by a corresponding decrease in the manifest range of accommodation. This point is well demonstrated in presbyopia.

It is also worth noting that a graduated scale for the amounts of paresis or paralysis can not be established as for presbyopia. Though, in a sense presbyopia is a paresis of the ciliary muscle according to Foster[38] and Kirk[39]. One reason is that the gradual recession of the near point and the latent range of accommodation to become manifest is a physiological process, progressing uniformly;[40] the other condition is a variable pathologic process, decidedly unequal.

In young people the amount of paresis can be approximately measured, but beyond 65 our present methods fail to establish whether the ciliary muscle is paretic or paralytic. Therefore ciliary paralysis is only of diagnostic import or becomes recognizable as a distinct manifestation in practically the "presbyopic." Then, too, only when the implication

[37]Presbyopia is a physiological lens hardening and never (so many say) due to any changes in the active portion of the accommodative mechanism. It is an open question whether an abnormal early hardening of the lens ever takes place, which can simulate presbyopia. It must be a hardness not due to any disease or complication.

[38]Foster, M. Text-book on Physiology, 1891, 6th ed., p. 47, says: "In presbyopia the failure or loss of accommodation may be due to a loss of the elasticity of the lens, or an increasing weakness in the ciliary muscle or to the parts becoming rigid."

[39]Kirk holds the identical view as Foster (Hand-book of Physiology, p. 71).

[40]One 25 years old with a normal near point can have as much ciliary paresis as a child of 10 or 15 whose manifest accommodation range is about one-half the normal and whose near point is correspondingly removed.

is greater than the latent range of accommodation does it annoy the patient.

As already said, a paralysis or paresis of the accommodation or of the ciliary muscle may yield the same clinical picture. Therefore, we must be careful in using this symptom as a diagnostic or prognostic point.

Mydriasis, complete or partial, frequently accompanies a paresis or paralysis of the ciliary muscle. Yet, as we have shown, one action is independent of the other. In a few instances a myosis was noted, with a paretic ciliary muscle[41].

In the peripheral paresis or paralysis, the ciliary body or processes are directly or indirectly involved[42]. The neuropathic involvments are frequently central, or peripheral while the myopathic are peripheral.

In general, the symptoms of a paralysis of the muscle of accommodation is dimetrically opposite to that of a spasm. The extent of the involvment depends upon whether the eye is emmetropic, brachymetropic, or hypermetropic; and, whether the patient is presbyopic or not. Paresis on the other hand simulates more closely what we term asthenopia, and often unable to be differentiated with our present day methods. The disturbances are greatest in the hypermetropic, as both near and far points are affected. Next comes the emmetrope, in him the near point is perhaps only farther removed than normal. The brachymetrope suffers the least, especially if the amount of error be 3 or 4 dioptres and no glasses were ever worn. In him, complete paralysis may take place and no subjective symptoms be complained of[43].

The annoyance is not very marked if the lesion is limited to one eye. A frequent accompaniment is micropsia. As already stated, other symptoms are often associated with this condition of the ciliary muscle, that we can provisionally tabulate them as follows:

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[41]This condition must be one form in which there is a central and a peripheral lesion.

[42]There may be a complete rupture of the zonula giving the same symptoms as a complete paralysis, yet the ciliary function is intact. Even vitreous disease has been reported to cause a relaxation of the zonula and resemble a paresis (Mauthner, Donders).

[43]This is particularly noticeable should a small amount of ciliary power be left. On account of the effort of the residual power of accommodation, the objects seem nearer and therefore smaller.

1. Accommodation alone affected—incomplete ophthalmoplegia[44] interna.

2. Accommodation and iris affected—complete ophthalmoplegia interna.

3. Accommodation, iris and external eye muscles—ophthalmoplegia totalis.

Any of these conditions may be a nuclear, a peripheral or an orbital lesion. The nuclear type of paralyses, especially of the progressive order, often have as their first symptom, a paralysis of the accommodation[45] (Mauthner, Graefe, and others). These nuclear affections may be bilateral or unilateral, complete or incomplete (vide foot note on ophthalmoplegia). The orbital lesions are more readily understood than the nuclear, as we may look upon them as peripheral in nature.

It is indeed difficult to explain how some constitutional or even brain lesion will practically isolate and only implicate the nuclear centre for accommodation. It can only be explained upon the basis of Huebner, Hensen and Voelckers. The nuclear paralysis or paresis may have the following brain lesions as its cause: hemorrhage, embolism, meningitis, thrombosis, tumor, abscess, cerebral syphilis, internal hydrocephalus, and posterior spinal sclerosis. Syphilis is by far the most frequent underlying cause for the nuclear implications. However, not in all of these is the entire centre of accommodation involved, unless a complete ophthalmoplegia ensues.

The ciliary muscle itself may be incapable of reacting in the proper proportion to the nerve stimulus and this would evidence itself as a paresis rather than a paralysis. This may be due to inflammation and its results. Senile changes in the ciliary muscle can produce a paresis. The constant inactivity of the ciliary muscle in the various forms of strabismus can give rise to a paresis or even a paralysis. Excessive nutritive

[44]The term ophthalmoplegia is, generic, having reference to the paralysis of any of the ocular muscles, intra- or extra-ocular. Mauthner (Die Lehre der Muskellaehmungen, p. 306-307) gives this very comprehensive classification:

Central or Peripheral	ophthalmoplegia	perfecta	unilateralis	exterior
			bilateralis	interior
		imperfecta	ibidem	ibidem

[45]The case of Heinrich Heine, the German Poet, is a celebrated case of progressive nuclear paralysis.

disturbances may also call forth a condition not unlike a paresis of the ciliary muscle.

The various conditions which may give rise to a paresis or a paralysis are as follows:

Syphilis.

Under this heading we may include tabes. The ciliary muscle is very often the first that suffers in tabes. The paresis, more often though a paralysis, is bilateral. Unilateral paralysis of the accommodation without mydriasis but with the characteristic anesthetic temple areas is also seen in tabes (Galezowski[46]). In the majority of the Argyll-Robertson pupils[47] will a paresis of accommodation be found. In simple confirmed syphilis, the accommodation implication is very late in manifesting itself, if at all. Brain syphilis frequently involves the accommodation centre very early, often being periodic and recurrent. This is not very strange when we consider the blood supply of this centre and accept the view of Adamkiewicz[48]. But why the centre for the intra- or extra-ocular muscles are so frequently involved in the early stage of syphilis can not be explained, unless it is due to their peculiar blood supply. Graefe says frequent recurring accommodation paresis with mydriasis, and alternating bilaterally, should lead one to suspect mental disease, especially if syphilis is present.

Usually the paralysis is bilateral and accompanied by either a partial or complete mydriasis. This is particularly true in tabes (Galezowski). Exceptions to this, however, are noted by Alexander, Kirmisson, Jeaffreson, Hosch, Ferron and Mauthner (vide special ref.) Hutchinson in his early reports (Med. Chir. Trans., 1878), mentions unequal in-

[46]He considers this condition almost pathognomonic of tabes.

[47]The Argyll-Robertson pupil is a nuclear lesion, often attributable to a hemorrhage. From here the lesion travels from the aquaeductus Sylvii to the floor of the third ventricle and destroys the connection between the third and first nerves without affecting the sphincter of the iris.

[48]Adamkiewicz has demonstrated that the sclerotic process in tabes is determined by the course of the arteries, especially in the brain. (The original is not accessible—quoted from Schmeichler, Knapp Archiv. Bd. XII. p. 335-364, 1883). The original article is: "Die Blutgefaesse des menschlichen Rueckenmarkes; Sitzungsb. der Academie der Wisschf. in Wien, 1882, Bd. IXXXIV.

volvement of the power accommodation in syphilitics. The specific involvement of accommodation is more apt to be central than peripheral and often depends on minute hemorrhages into the nucleus. This condition has been demonstrated post-mortem in a number of cases. All the sudden ophthalmoplegias with recurrences are indicative of syphilitic nuclear lesions, rather than peripheral.

Inherited syphilis does not as frequently cause a paralysis as the acquired form (St. Bernheimer, gen. ref. No. 21, p. 14). A latent form of syphilis can be recognized by a paralysis of accommodation with pupillary differences or bilateral mydriasis. These latter two conditions are often for a long time the only expression of a syphilitic infection. However, syphilis, especially of the nervous system, is more prone to involve the extra-ocular muscles than the intra-ocular, giving an ophthalmoplegia externa (complete or incomplete).

J. Hutchinson's writings show very clearly that the ciliary muscle implication is very rare in inherited syphilis.

The prognosis in this class of cases is not very favorable or even encouraging. Alexander (vide special ref.) does not believe a cure ever takes place.

Sympathetic Ophthalmia.

In this direful disease ciliary muscle paralysis may be a very early manifestation in the sympathizing eye. Fuchs and Cuignet lay considerable stress upon its being fairly diagnostic, providing conditions for sympathetic ophthalmia are present. The writer can affirm this observation in a case which he had under treatment. How this ciliary paralysis is brought about is a question. Randolph's researches on the influence of toxines in inflammations of the eye are of paramount interest in this connection. Whether it is a peripheral or a central paralysis has not been stated. The writer considers it a pure peripheral lesion, as are so many of the toxic type.

Perhaps it is a neuropathic paralysis. The mydriasis, which often is of a moderate degree, is likewise a peripheral lesion. In sympathetic irritation this ciliary complication has not as yet been reported, hence in doubtful cases this symptom will prove of some value.

Local Peripheral Causes.

The paresis or paralysis here are usually secondary to some disease in the iris, ciliary body, choroid, or retina. If it follows one of the mentioned causes it is of the myopathic type, and it is then the result of plastic exudates or organized inflammatory material binding down the ciliary body and processes. Atrophy of the muscle is then prone to follow these changes. It is not a want in the nerve stimulus, but an inability on part of the muscle to contract. This condition may account for the reduced vision in such cases that still have clear media and no other complications to explain the reduction.

Glaucoma frequently produces a paralysis or paresis of the accommodation. More often it is a paresis. We are all familiar with this symptom, and often have it as a premonitory sign, recognizable by the frequent desire to change the "glasses." This condition is more often witnessed in that form of glaucoma wherein we have a shallow anterior chamber and a markedly dilated pupil. However, it is quite evident that the passive part of the accommodative mechanism suffers to a certain extent as well.

Injuries in the immediate vicinity of the eye frequently call forth a paralysis of the accommodation. Injuries to the cranium may yield nuclear accommodative interferences, with more of a general ophthalmoplegia. Very often there is a complete rupture of the zonula, which will cause a passive paralysis, as the ciliary muscle is left intact. This form of paralysis is not rare, as is evidenced by Power, Harlan[49] and others.

Defective teeth are also responsible for ciliary paresis. Schmidt-Rimpler and Schmidt (1868) have cited a series of cases of asthenopia of the paretic type that were due to defective upper teeth. This is a peripheral reflex paresis. Schmidt[50] is inclined to think it very common indeed. Few cases were observed by the writer. No recent reports are at hand. Linnell[51] (1884) reports a case of paretic ciliary accommodation as being due to an irritation of the superior

[49]Harlan and Power. Vide special ref.

[50]Schmidt. Vide special ref. He found 73 cases in a series of 92.

[51]Linnell. Vide special ref.

maxillary nerve. This must be the basis for the other cases as well.[52]

Over-exertion of any description is a fruitful cause for paresis of the ciliary muscle. It is peripheral and perhaps many of the accommodative asthenopias come under this heading. Any strenuous occupation or the so-called misuse of the eyes may give rise to it. Panas[53] mentions a case of unilateral paralysis with amblyopia as the result of undue exposure to intense electric light. Working over bright fires seem to cause it also (Colsman).[54] Mydriasis as a rule does not accompany this form. Students, typewriters, and electricians form a large contingency.

This variety is mainly due to the accumulation of waste energy material. Hutchinson's[55] cases (v. special ref.) following shock and lactation will properly belong to this category.

Poisons, either local or general, very often produce paresis or paralysis. As to the local ones, we need only mention the cycloplegics and their allied group of remedies. It is often a very important symptom in meat, fish or vegetable poisonings. It is more in the nature of a marked paresis than an outright paralysis. Mydriasis is often an associated symptom. The agent is invariably a ptomain and acts peripherally, in a manner similar to the cycloplegics. Therefore it is apt to be a nerve paralysis. Very often it is the first symptom which manifests itself in these cases. A like condition has been noted in tyrotoxicon poisoning (Fisher, Fuerst, Weiss, Leber, Knies, Groenouw, vide special ref.). In this particular we must not forget to mention the tobacco, alcohol, cocaine and morphine habits. In alcohol it is a paresis from the beginning, while in tobacco, it is a spasm first, which later is followed by the paralysis. In cocaine, we have a paresis followed by a complete paralysis.

Let us now consider some of the diseases such as diabetes, diphtheria and the like with reference to ciliary paralysis and paresis.

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[52]The disease travels by way of the spheno-palatine and ciliary ganglion, the connecting link being a small nerve fibre.

[53]Panas. Vide ref.

[54]Colsman. This is a very unique case. Vide ref. No. 9.

[55]Schapringer's case of paresis with apparent myopia may be of this class, though he does not say so, yet the history points to it.

Diabetes[56].

Very frequently diabetes involves the accommodation apparatus[57]. Whether this involvment is central, peripheral, or due to the accompanying reduced muscular vitality which is common to diabetics, is still a debated question. No doubt we can accept all three causes at times. It is usually a paresis and seldom associated with mydriasis. Von Graefe first called attention to this lesion in 1858 and since then many observers have recorded cases. Often it is among the first symptoms. Many regard it as a toxic manifestation and the writer shares the same view. For, upon the decrease in the glycosuria the intra-ocular paresis rapidly improves[58]. Jacobson looks upon the paresis as a peripheral neuritis or as a hemorrhage. Foerster, as many others, regards it as due to the general muscular weakness. It may be a nuclear lesion and then unilateral (perhaps a hemorrhage). The perverted presbyopia must not only be looked upon as a ciliary paresis but also as due to changes in the lens itself.

Influenza[59].

The interference of accommodation was among the first "so-called out of the ordinary symptoms" recorded. At times the paresis or paralysis, as in diphtheria, appears some time after the patient is entirely well. Very often though, it comes on during the progress of the attack. Rarely does it appear as a premonitory symptom. The disease being of germ origin, we can well attribute the accommodation interference of some toxin, just as in diphtheria. It apparently is of a peripheral type; when brain complications arise early in the disease, then nuclear implications are prone to occur.

(*Continued next issue.*)

[56]Berger, E; B. first advanced the toxic theory in diabetes, not only of the intra, but also of the extra-ocular muscle affection. He assigns his reasons in a very valuable contribution (vide ref.).

[57]Galezowski. In 1883, in a thesis, he says 7 per cent. of all diabetics have some muscle paralysis. No doubt some are nuclear hemorrhages.

[58]Knies. K. favors the acute intoxication theory. (It does seem a rational view both here and in albuminuria). See note on Berger.

[59]Williams. R. A. (v. special ref.) gives a detailed account of paralysis and paresis in influenza, with a review of facts. Statistics are very meager, but would not be, were the cases reported.